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ARCHIVES OF PEDIATRICS

A MONTHLY DEVOTED TO THE
DISEASES OF INFANTS AND CHILDREN

JOHN FITCH LANDON, M.D., Editor

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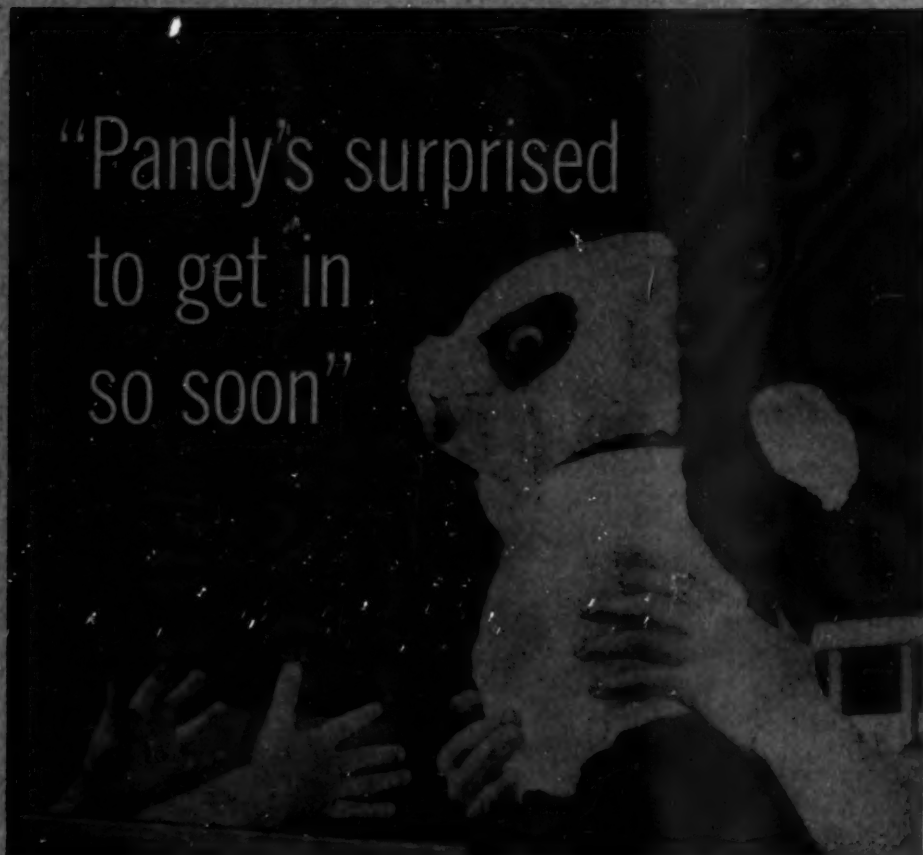
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1. Fox, C. L., Jr., et al.: An Electrolyte Solution Addressing Plasma Concentrations with Increased Potassium for Patients That are Electrolyte Depleted. *J. A. M. A.*, March 2, 1972.

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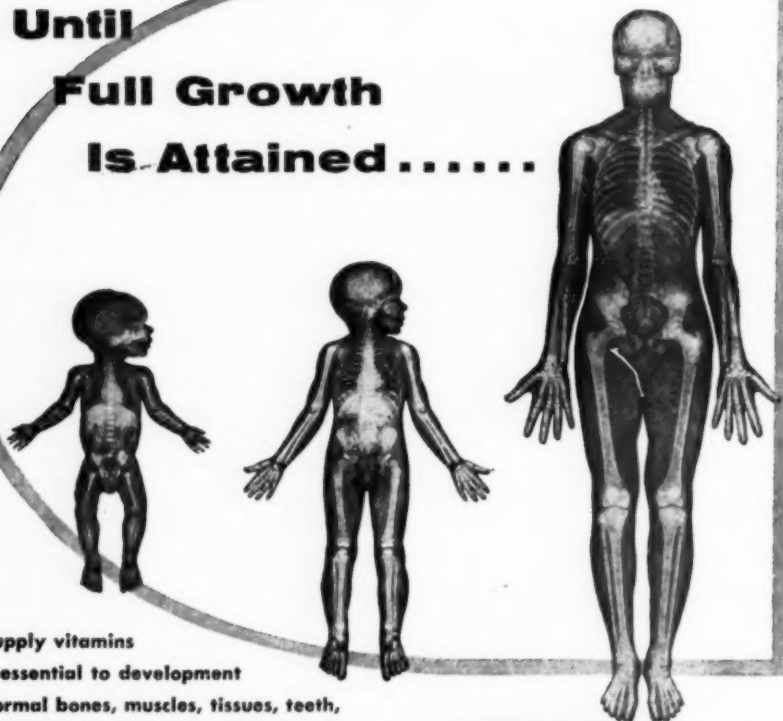
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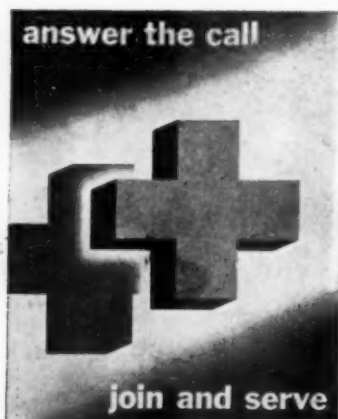
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(b) *Peel oil content significantly lower:* Sam-

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(c) *Bacterial counts dramatically lower:* Bacterial counts were found to be as high as 350,000 per ml. in home-squeezed samples, but were uniformly low in MINUTE MAID.

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New Assays Reaffirm Dietary Advantages of Minute Maid Fresh-Frozen Orange Juice on a Cost Basis

A second report comparing the individual mineral and vitamin values of MINUTE MAID Fresh-Frozen Orange Juice and home-squeezed juice of the same type oranges has recently been published.

In this latest study, each sample was analyzed separately. The analyses showed that MINUTE MAID Fresh-Frozen Orange Juice was equal to, or superior to, the home-squeezed juice in all of the components listed below:

TABLE
Mean Values in Samples Tested

COMPONENT	UNITS	MINUTE MAID FRESH-FROZEN ORANGE JUICE	HOME- SQUEEZED ORANGE JUICE
Betaine	mg./100 ml.	40	46
Biotin	mcg./100 ml.	0.26	0.26
Choline	mg./100 ml.	12	12
Calsalt	mg./100 ml.	74	67
Folic acid	mcg./100 ml.	3.2	3.2
Iodine	mcg./100 ml.	0.24	0.21
Manganese	mcg./100 ml.	33	18
Nitrogen			
Total	mg./100 ml.	104	79
Amino	mg./100 ml.	22	22
Volatile	mg./100 ml.	8	7
Non-volatile	mg./100 ml.	96	72
Pantothenic acid	mcg./100 ml.	146	145
Para-aminobenzoic acid	mcg./100 ml.	4	4
Phosphorus	mg./100 ml.	19	18
Potassium	mg./100 ml.	380	200
Riboflavin	mg./100 ml.	18	17
Tryptophan	mg./100 ml.	107	104
Vitamin A	mcg./100 ml.	19	16
Thiamine	mcg./100 ml.	87	83
Vitamin B ₁₂	mcg./100 ml.	0.0022	0.0012

Although the results are again susceptible to variation according to crop and year, Fresh-Frozen MINUTE MAID was equal to the home-squeezed juice in the samples tested for the largest number of components listed; and in the mean values for iodine, manganese,

potassium, Vitamins A and B₁₂, MINUTE MAID showed appreciably higher values.

SUMMARY

These new findings help enlarge professional knowledge of the nutrient constituents of orange juice in general and add fresh evidence that, on a cost basis, MINUTE MAID Fresh-Frozen Orange Juice has significant dietary advantages. Penny for penny, MINUTE MAID offers not only more Vitamin C, but also more of all the other vitamins and minerals listed than home-squeezed orange juice.

Taken in conjunction with the previously published findings, this should confirm the choice of physicians who recommend MINUTE MAID in place of home-squeezed orange juice.

REFERENCES:

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- (2) Joslin, C. L., and Bradley, J. E., Journal of Pediatrics, Vol. 39, No. 3, pp. 325-329 (1951).
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- (4) Assn. Official Agricultural Chemists: Methods of Analysis, 7th ed. Washington: Assn. Off. Agric. Chemists, 1950.



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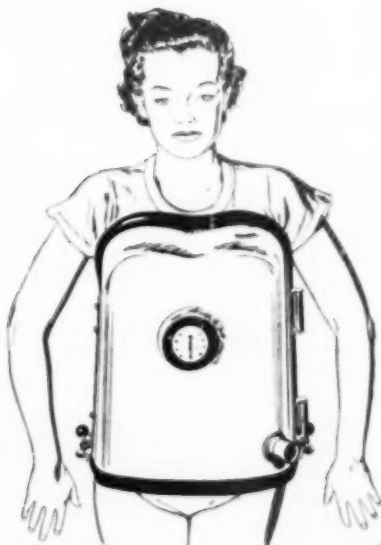
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MONGOLISM AND "MISSED MONGOLISM" FOLLOWING MATERNAL ILLNESS

EDWARD E. BROWN, M.D., F.A.A.P.

Ashland, Ore.

This is a report of maternal illnesses present early in pregnancies which resulted in the birth of seven mongols (Figs. 1-7) and four "missed mongols" (Figs. 8-11).

The term "missed mongol" or "forme fruste" is used here to describe four patients who bore a degree of resemblance to mongols and who had congenital defects similar to those often present in mongols. As in true mongols, three other characteristics were usually present in this group—premature birth, subnormal birth weight and retarded mentality.

The birth of a mongol may follow many types of maternal illness during early pregnancy.¹ In 1908, Hill reported that in 4 out of 8 cases of mongolism, history revealed bad health in the mother; he concluded that there was "the presence of some depressing influence during the gestation period of the mother." Brousseau² noted, among 379 cases of mongolism, ill health in the mother in 179 or 47.2 per cent. Bleeding during pregnancy was noted by Schröder³ and Beidleman⁴ in approximately 20 per cent of mothers who subsequently gave birth to a mongoloid child. Benda⁵ also observed cases where menstruation continued during the first three months of pregnancy, "an indication that something was wrong with the mechanism of pregnancy." The history obtained by Ingalls and Davies revealed that mothers were sick throughout pregnancy;



Fig. 1.



Fig. 2.



Fig. 3.



Fig. 4.



Fig. 5.



Fig. 6.



Fig. 7.



Fig. 8.



Fig. 9.



Fig. 10.



Fig. 11.

vomiting was a prominent symptom.⁶ Recently, Plummer⁷ recorded the birth of two mongols and six microcephalics with mental retardation among 205 women exposed to the atomic bomb blast in Hiroshima. These women were in the first twenty weeks of gestation and many of them showed subsequent evidence of radiation illness.

With increasing frequency mongolism is being noted after virus and streptococcal diseases attacking the mother, usually in early pregnancy. Virus diseases include rubella,⁸⁻¹³ measles,¹⁴ mumps,^{8, 10, 15} and influenza.^{8, 10} Among the presumably streptococcal diseases are mastoiditis, pleurisy, otitis media, sinusitis and nephritis.^{8, 10, 16}

The eleven mongols and "missed mongols" under my observation were selected only on the basis of a reliable history. It is noteworthy that ten of the eleven revealed some history of maternal illness during the first three "critical" months of pregnancy. Maternal illnesses included toxemia and severe vomiting in seven. Other illnesses present during early pregnancy, prior to the birth of our eleven patients, are shown in Table 1. As a control observation, I inquired into the maternal history of the last eleven consecutive normal babies; except for nausea, there was no first-trimester illness in all eleven.

DISCUSSION

While the number of cases is small, the existence of some maternal abnormality in ten of the eleven consecutive cases, and none

TABLE 1. Data on Eleven Mongols and "Missed Mongols" Including History during Pregnancy

Case Number	Age In Years	Maternal Illness During Pregnancy	Birth Weight	Estimated Prematurity In Months	Other Data Regarding Patient and Siblings
MONGOLS					
1. (Fig. 1)	1½	Vomiting first 7 months; rheumatic pains throughout	5 lbs. 15 oz.	½	Barely able to sit up
2. (Fig. 2)	2	Vomiting throughout	3 lbs. 14 oz.	2	Unable to walk
3. (Fig. 3)	2	Vomiting first 3 months	5 lbs. 4 oz.	0	Unable to walk; congenital glaucoma; umbilical hernia
4. (Fig. 4)	4	"Constant cold," active chronic sinusitis; recurring rheumatic pains	7 lbs.	1	Birth weight of older brother, 7 lbs. 10 oz.; younger sister, 8 lbs. 2 oz.
5. (Fig. 5)	4½	Vomiting first 6 months; "one cold after another"	7 lbs. 7 oz.	½	Walked at 2 years, 4 months; brother, aged 1, normal.
6. (Fig. 6)	27	Influenza second month	12 lbs.	0	Brother, aged 24, normal
7. (Fig. 7)	17	Vomiting throughout; nephritis	7 lbs. 9 oz.	0	Patient is third and last child. Birth weight of two older siblings: 11 lbs. 6 oz.; 12 lbs. 3 oz.
"MISSED MONGOLS"					
8. (Fig. 8)	5	Mother Rh negative; father Rh positive; no maternal illness	8 lbs.	0	Patient is deaf, has spastic gait and marked mental retardation. Birth weight of older normal sibling, 9 lbs. 5 oz.; second pregnancy resulted in stillbirth
9. (Fig. 9)	5	Vomiting and rheumatic pains recurring throughout	6 lbs. 3 oz.	½	Partial deafness, imperfect speech, large furrowed tongue, slight mental retardation
10. (Fig. 10)	10	Vomiting first 3 months	5 lbs. 9 oz.	1	Patient interventricular septum, club feet at birth; pneumonia 5 times; marked mental retardation. Sister, aged 5, normal
11. (Fig. 11)	7	Active sinusitis, infectious ashlma and cough throughout	5 lbs. 8 oz.	1	Patient walked at age 2, when inguinal hernia was discovered; right internal strabismus; slight mental retardation. Normal siblings, aged 16 and 10

in the controls, seems provocative. Innumerable women have toxemia without giving birth to mongoloid children, but most toxemias occur later in pregnancy. It should be of future interest to note the effect on the infant of toxemias and other illnesses during the first trimester.

CONCLUSIONS

Among eleven mongols and "missed mongols" reported in this study, history in ten revealed some type of maternal illness during the first three months of pregnancy. There was vomiting in seven mothers, one of whom also had nephritis and another had rheumatic pains. Two mothers had severe sinusitis, one with an associated bacterial asthma and another with rheumatic pains. One mother had influenza in the second month of pregnancy. The eleventh case was one of Rh incompatibility, but without maternal illness.

Viral and bacterial diseases during the first three months of pregnancy may be a more common cause of mongolism than has been recognized previously. Further studies of the health of mothers during the first trimester of pregnancy are needed to verify this suspected relationship.

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PANHAEMATOCYTOPENIA

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Depression of the cellular elements of the blood, in whole or in part, may occur in many conditions. Perhaps the best known of these is "aplastic anaemia," where reduction of the red cells, leucocytes and platelets in the peripheral blood is found together with aplasia of the bone marrow. More recent studies of cases of aplastic anaemia have, however, shown that the bone marrow may be normally cellular or even hyperplastic¹; such cases usually run a sub-acute or chronic course and are more often found in adults 25 to 50 years of age rather than in the younger age groups. In still other cases of "aplastic anaemia," there may be no abnormality in the leucocytes or platelets, and even occasional nucleated red cells, as well as polychromatophilia and mild reticulocytosis (as much as 5 per cent in rare cases), may be found.

It has been known for a long time that certain diseases affecting the liver and/or spleen, such as cirrhosis of the liver or congestive splenomegaly (Banti's syndrome), may be accompanied by depression of one or all of the elements of the blood. These vague and diverse problems have been clarified in 1946 by Doan² and Damashek,³ the former speaking of the syndrome as panhaematocytopenia and the latter as hypersplenism. The syndrome may occur in a partial way, where depression of only one element of the blood to the exclusion of the others occurs, or in a total complete form, where all the formed elements of the blood are reduced. Hypersplenism and haematocytopenia may be primary and idiopathic or secondary to such diseases as rheumatoid arthritis (Felty's syndrome) lipoidoses, syphilis, sarcoidosis, malaria, Hodgkin's disease etc. As a designation to this syndrome, hypersplenism is a most appropriate term as it stresses the usually spectacular benefit of splenectomy.

The diagnosis of hypersplenism is based on three criteria⁴:

I. Cytopenia, anaemia, granulocytopenia and thrombocytopenia.

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II. Palpable enlargement of the spleen; this however is not invariable in thrombocytopenic purpura.

III. An active bone marrow showing good production of the types of cell that are diminished in the circulating blood.

The syndromes of primary hypersplenism are⁴: 1) Spherocytic anaemia (congenital haemolytic icterus). 2) Splenic thrombocytopenia (thrombocytopenic purpura). 3) Splenic neutropenia. 4) Splenic pancytopenia: a) with haemolytic anaemia, b) with non-haemolytic anaemia.

Primary hypersplenism, apart from spherocytic anaemia and thrombocytopenic purpura, is not common. Out of 270 splenectomies for hypersplenic syndromes in 17 years, Doan⁵ reported only 13 cases for primary splenic neutropenia and 11 for primary splenic pancytopenia. Welch and Dameshek⁶ found 20 cases only of primary splenic pancytopenia out of 220 splenectomies in ten years.

The mechanism whereby the spleen, by excessive activity, brings about this result is by no means established. Two, not necessarily exclusive, theories are recognized at present. Doan and his co-workers believe that in hypersplenism, the phagocytic activity of the spleen for cells is exaggerated; the peripheral paucity of these cells would be due to phagocytosis in the spleen while the hyperplasia of the marrow would be a physiological compensatory mechanism. In favour of this theory, they cite the demonstration of cellular phagocytosis in the spleen, on microscopic examination, as well as the squeezing of the cells as a result of the splenic contraction that follows the injection of 0.5-1 ml. of adrenalin.

Dameshek, on the other hand, attributes to the phagocytic activity of the spleen a minor role. Instead, he ascribes to the spleen a regulating influence on the bone marrow, in that it controls the delivery of red cells, granulocytes and platelets from the marrow and possibly their formation as well. In hypersplenism these two factors are inhibited and the marrow is therefor full of cells that are not being delivered to the blood; the proper development of some elements—notably the platelets—may be inhibited. In favour of this theory, Dameshek cites the fact that in thrombocytopenic purpura, the platelet count sometimes rises an hour or so after ligation of the splenic vessels; this is more easily explained by release of inhibition rather than by a sudden cessation of phago-

cytosis. He also regards the adrenalin test as of no diagnostic value, as it often fails to produce the expected results.

In his book on spleen and hypersplenism, Dameshek⁷ has described three cases of primary panhaematocytopenia, one of which had a very large spleen with evidence of haemolysis together with depression of all corpuscular constituents of peripheral blood; the other two cases showed the picture of "aplastic anaemia" in the peripheral blood and were associated with an active bone marrow but no signs of haemolysis. The ages of these three cases were 55, 57 and 47 years.

We have recently come across two examples of panhaematocytopenia in children that illustrate the two subgroups.

CASE REPORTS

Case I. K. M. (Hosp. No. 728/53): a girl, 12-years-old, was admitted to the hospital for progressive lassitude and pallor, spots on the skin, bleeding from the mouth and nose, recurrent fever and diarrhoea of one month duration. One year ago, she had a swelling of the neck (haematoma?) that subsided after some time. Past history irrelevant; no history of drug intake.

Examination showed a very pale girl with buccal and faucial ulceration. Cervical, axillary and inguinal glands very slightly enlarged; spleen not felt. Heart a little dilated with a functional haemic murmur over the apex and pulmonary area. Liver, felt one cm. below costal margin in midclavicular line. Hemoglobin 20 per cent, R.B.C. 2.2 million per cmm., W.B.C. 6 thousand per cmm., polymorphs 8 per cent, lymphocytes 90 per cent, eosinophils 1 per cent and monocytes 1 per cent. Platelets, 11 thousand per cmm., bleeding time over 10 minutes; icterus index 4. The anaemia was rapidly increasing in spite of repeated transfusions; hemoglobin reached 16 per cent and R.B.C. 700 thousand per cmm. on the 24th. day of hospitalization. No immature cells or reticulocytes were found in the peripheral blood. X-ray of bones showed no abnormality. Marrow biopsy showed a cellular marrow with abundance of myelocytes. The patient received blood transfusion on five occasions, two of which were of packed R.B.Cs. She died thirty days after admission.

Autopsy revealed subepicardial and subendocardial haemor-

rhages in the heart, cortical punctate haemorrhages in the cerebellum, a red marrow, oedema of the lungs, and membranous colitis affecting the caecum and hepatic flexure.

Case II. A. Z. (Hosp. No. 3225/52); a boy, 14-years-old, complained of pallor, weakness, repeated attacks of epistaxis and abdominal swelling for the last three years. Apart from an old urinary bilharziasis that was treated, his past and family history was irrelevant. Examination showed a marked degree of jaundice, moderate clubbing of the nails, a huge spleen reaching below the umbilicus with a bunch of dilated vessels in the abdominal wall at its lower pole over which a thrill was felt. The liver edge was felt two centimeters below the costal margin in the midclavicular line. No ascites could be detected. The heart was dilated with a systolic murmur over the apex and pulmonary area, lungs free. During hospitalization, hemoglobin ranged from 30-60 per cent and R.B.C. from 1.620 to 3 million per cmm., W.B.C. from 2.5 to 4 thousand per cmm. (polys. 60-80 per cent, lymphs 14-40 per cent, eosinophils 2 per cent), reticulocytes 6-14 per cent, nucleated red cells 4 per cent and platelets 120-190 thousand per cmm. Coagulation time 7 minutes, bleeding time 2 minutes and fragility 0.45-0.35 per cent. Icterus index varied between 12 and 100. Urine showed increased urobilinogen but no bilirubin. Blood film for malaria negative. No bilharzia ova were found in the urine or stools. Sigmoidoscopy showed some hyperaemia of the mucous membrane but no evidence of bilharziasis. Blood proteins 4.7 gms. per 100 ml. (albumin 3.2 gms. per 100 ml.). Alkaline phosphatase 9 units (King-Armstrong), thymol turbidity 4+. Radiography of the bones, heart and lungs was normal. No oesophageal varices could be visualized by a barium swallow. The bone marrow showed the following picture: myeloblasts 0.5 per cent, myelocytes 27.5 per cent, juveniles 12.5 per cent, staff 15.5 per cent, segmented 7 per cent, eosinophils 3.5 per cent, baso. 0.0 per cent, lymph. 5 per cent, macro. 6 per cent, normo. 21.5 per cent and the myeloid erythroid ratio 7/3.

Splenectomy was done three months after admission and the microscopic picture of the spleen was examined. The pathologist reported: "The splenic pulp shows marked fibrosis and is infiltrated by numerous mononuclears and several eosinophils. The

malpighian bodies are atrophied with inactive germ centres. Few of the histiocytes contain dark brown pigment. The appearances are compatible with those of endemic splenomegaly."

After the operation, and for four months now, the blood picture has been found repeatedly to be almost normal. Hemoglobin ranged from 60-68 per cent and R.B.C. from 2.550 to 4.250 million per cmm., W.B.C. from 10.4 to 16 thousand per cmm. (polys. 45-80 per cent, lymph. 20-40 per cent, eosinophils 0-15 per cent), reticulocytes 1 per cent, and platelets from 280-500 thousand per cmm. Icterus index 5 units. Urine showed neither bilirubin nor increased urobilinogen excretion.

COMMENT

The first case reported was provisionally diagnosed as aplastic anaemia. This was readily excluded by demonstrating a hyperplastic bone marrow, for, though such finding may occur in aplastic anaemia, the patients affected are usually in the older age groups. Some authors indeed stress the fact that, although the marrow in aplastic anaemia may be hyperplastic, such hyperplasia is found only in scattered and isolated foci. Others go so far as to consider any case with hyperplasia of the bone marrow as an instance of hypersplenism. Aleukaemic leukaemia was also excluded by the result of bone marrow puncture as well as by the absence of leukaemic infiltrations at autopsy.

The second case presented a haemolytic type of anaemia and jaundice. Congenital spherocytic anaemia was excluded on the basis of the negative family history, the morphology of the red cells and the result of the fragility tests taken together. The case is one of panhaematocytopenia with haemolytic anaemia. Except for an old history of treated bilharziasis, no evidence of any associated disease was found and so it seems that the panhaematocytopenia is primary. On the other hand, the pathological report on the spleen revealed appearances consistent with endemic splenomegaly. It is thus possible that the panhaematocytopenia is secondary to bilharziasis. As far as the literature available to us, this would be the first case of its kind to be reported.

Although the syndrome of panhaematocytopenia and hypersplenism has only recently been clarified, and although the cases

of primary panhaematocytopenia described since its clarification have been few, the condition must always be borne in mind. This is mainly because of the usually good results of splenectomy and the otherwise downhill course of the cases not so treated. The return of blood picture to normal, the disappearance of jaundice—both clinically and by laboratory tests—as well as the great improvement in the general condition of our second case illustrate the value of splenectomy in such cases, while the downhill course and the fatal issue in our first case is typical of the untreated disease. Whatever the pathogenesis of panhaematocytopenia, there is an agreement on the high success that attends the removal of the spleen and the relatively low mortality of the operation. Dameshek has given a success of 80 per cent among his 26 splenectomised cases of haematocytopenia of different types (primary or secondary, partial or total), including three of the primary panhaematocytopenic type which he described in his book. The mortality in his series was less than 5 per cent. Doan, though entertaining different views on the pathogenesis of the condition, does agree on the value of splenectomy in most of these cases.

SUMMARY AND CONCLUSIONS

The subject of hypersplenism (panhaematocytopenia) is briefly reviewed.

Two additional cases are reported.

The first case was one of total primary panhaematocytopenia with a nonhaemolytic anaemia that ended fatally. The spleen could not be felt at any time during the patient's hospital stay.

The second case was one of total haematocytopenia with a haemolytic anaemia. The spleen was huge and splenectomy effected a cure. The panhaematocytopenia is possibly secondary to an old bilharzial infection—though evidence of this could not be obtained before operation.

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THE EMOTIONAL PROBLEMS OF THE ILLEGITIMATE CHILD

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The child who is born into this world without his parents being married is stigmatized from the very beginning, not because of any fault of his own, but because of the indiscriminate action of his parents, and because society does not accept children born out of wedlock. The illegitimate child has many difficulties to face and overcome simply because of the nature of his origin.

It is now generally conceded that for the normal development of a child, the presence and influence of both his parents is equally necessary. While the need for the mother is peremptory and immediate, the father does not enter into the child's life until consciousness and awareness develop for the meaning of home and family to be grasped. When this stage is reached, the absence of the father, or suitable father substitute, leaves the child without an important balancing influence. When the child reaches an age of awareness he has a need for authority. He needs to respect and obey, to be told what is right and what is wrong. The boy needs to look up to the father and aspire to become like his father. The father should be a pillar of strength and a hero-ideal for children of both sexes.

In the case of illegitimate birth, the child's reactions to life are bound to be not completely normal. To be fatherless is difficult enough, but to be fatherless with the stigmata of illegitimate birth is quite unbearable. He feels himself to be under a constant handicap not of his own doing and about which he can do nothing. He feels himself to be the victim of an injustice beyond his control.

The illegitimate child feels out of place in the social scheme of things. He feels that he does not belong. The child's quest is to be accepted, to belong unquestionably. Emotional satisfaction is derived when the child has an undoubted feeling that he is part of the world about him. The child has an inborn capacity to relate to the father by identification and experience the emotional things of life which make us all human beings, capable of loving and of being loved, and of taking an active or creative part in social experiences. The illegitimate child is usually denied this.

The child born out of wedlock in time develops feelings of inferiority, inadequacy and shame, together with feelings of envy and jealousy. He is extremely jealous of children who have the love and protection of both parents, while he lacks these. He feels insecure and isolated. He feels insecure because he finds himself in a situation different from that of his playmates.

The illegitimate child feels most acutely that he is not loved and appreciated as other children are. He feels that he is living in a vacuum, alone, unwanted, rejected. He lacks love and he does not know how to give love.

For the child born out of wedlock, love is required to give him a feeling of being wanted and of belonging. The child needs love even more than the adult because he is more helpless and more plastic. Only by experiencing love can he learn what love is and knowing love, return it and experience emotional satisfaction. When the illegitimate child has little love, he has of necessity to turn his love to himself. When this is the case he cannot acquire the habit of giving love to others, a socializing bond to his environment which is so important for his proper emotional development.

Quite often the child born out of wedlock becomes neurotic for the reason that he is denied love. He acquires peculiar mannerisms and behavior patterns. He becomes resentful and, in more extreme cases, delinquent.

For the illegitimate child to be loved means to be protected against anxiety, to have self-esteem and to feel that he has a place in society. Love to him means abolishing loneliness and the dreadful feeling of isolation, of not belonging to anyone, of living in an emotional vacuum. Love means a merging of the one who loves with the one who is loved, so that the self of one person includes the self of the other, that which is one's is also the other. As a consequence of such merging there is a mutual and complete participation in various feelings. Love is the intimate togetherness of the parent and the child in the experience of we-ness (being one).

Love in the case of the illegitimate child has a widening and broadening influence on his personality development. It permeates his thoughts and actions. It gives an added dimension to his everyday life by its fulfilled restfulness.

In addition to love, the emotional care of the child, born out of

wedlock, requires a building up of his self-esteem and an awareness of himself as a worth-while individual. He must be so managed that he builds up an alive interest and awareness in everything and everyone. He must be given a mature and independent outlook. He must be made not to feel in constant need of protection. It should not be necessary for him to crave a constant demonstration that he is loved. He must be taught to tolerate disappointment.

Such a child should be taught that all his desires cannot be gratified at all times. He must find his greatest joy in being useful and helpful. He must be taught to get along with other people and not be unduly demanding. He must be able to meet his disappointments without attacks of rage, tantrums and sulking.

For the illegitimate child mental health means the avoidance of unnecessary conflict. To avoid conflict means to be well-adjusted within himself and to his surroundings. He must learn to absorb emotional shocks. When this is done he can live with some degree of contentment and happiness.

CONGENITAL DEFECTS DUE TO RUBELLA. (*Revista Espanola de Pediatria*, Zaragoza, 9:135, Jan.-Feb. 1953). Two cases of congenital defects due to rubella are reported. Both infants were born to normal couples who had no familial history of malformations or of congenital cataract. The mother of the first infant had rubella in the first two weeks of her first pregnancy. The infant was seen at the age of 2½ months, and a diagnosis of unilateral congenital cataract of the central type was made. The infant was otherwise normal and had normal hearing. The mother of the second infant had the rubella in the sixth week of her third pregnancy. Her other two children are normal. The infant was seen at the age of 3 days. A diagnosis of bilateral congenital cataract of the central type and of congenital heart disease was made. The nature of the infant's heart disease has not been determined. His hearing is normal. The author advises that gamma globulin be given to a woman who is exposed to rubella in the first trimester of pregnancy. The prophylactic action of the drug lasts 30 days. If any further contact occurs after 30 days, the treatment should be repeated. Induced or natural rubella immunization is not effective. —*Journal A.M.A.*

PARA-AMINOBENZOIC ACID AS A FUNGICIDE FOR COCCIDIOIDES IMMITIS*

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Para-Aminobenzoic acid has been used for local therapy in ring-worm infections and in rickettsiae infections, such as Rocky Mountain Spotted Fever. The drug is rapidly absorbed and excreted, and its concentration can be followed easily by blood tests. These ideal characteristics stimulated us to investigate with in vitro studies to determine if it would inhibit the growth of *coccidioides immitis*.

Another drug used in fungi infections called ethyl vanillate had a benzoic radical in its formula. It was used by Christie¹ in disseminated histoplasmosis and by Cohen² in disseminated *coccidioides*.

Various dilutions of p-aminobenzoic acid were mixed into petri plates containing Sabouraud's media. Each plate was inoculated with a loopful of pus containing spherules from an active coccidioidal granuloma, then sealed and set at room temperature. The plates were read daily after two days of incubating.

The controls showed the fungi in large quantities. Complete inhibition of *coccidioides* was manifested in the plate with 1,500 micrograms per milliliter and fungistatic at 1,000 micrograms per milliliter. The same in vitro study was done with the mycelia from a coccidioidal meningitis case and complete inhibition was noted at 1,500 micrograms per milliliter. Other studies showed complete inhibition at 1,000 micrograms per milliliter using the spherules as the inoculum. However, 1,500 micrograms per milliliter is the most conservative concentration in the over-all study.

Ravenel³ showed that large concentrations were possible in Rocky Mountain Spotted Fever and thus this in vitro study will allow us, or any interested party, to give it a clinical trial on the disseminated coccidioidal cases.

CONCLUSION

Para-Aminobenzoic acid has been noted to completely inhibit

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the growth of *coccidioides immitis* at 1,500 micrograms. Its properties allow it to be used now for a clinical trial in the disseminated cases of *coccidioides*.

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TREATMENT OF TUBERCULOUS MENINGITIS. (Nederlandsch Tijdschrift v. Geneeskunde, Amsterdam, 96 : 2479, Oct. 4, 1952). At the children's clinic of the University of Utrecht a total of 61 children with tuberculous meningitis were treated with streptomycin between 1947 and 1951. Of the 30 children who were treated throughout the entire course of the disease at this clinic, 14 died and 16 are alive. Of the 31 children who had received their initial treatment elsewhere, 20 have died. At the university clinic a combination of streptomycin and calcium chloride was given both intramuscularly and intrathecally. In the patients in whom treatment was effective, clinical manifestations and changes in the cerebrospinal fluid usually disappeared from 9 to 12 months after onset of treatment. Those in whom this was not the case usually died. It was found that chronic tuberculous meningitis assumes one of two distinct forms. In the first form there is continuous loss of consciousness, with extensive neurological changes and terminal hyperpyrexia. In the second form the patients remain conscious until shortly before death, but they have severe convulsions and vomiting and become cachectic; their temperature is usually normal. Postmortem examination reveals in those with the first form severe hydrocephalus with signs of recovery in the meninges; those with the second form show extensive basal meningitis. The authors feel that the intraventricular pressure should be determined in all cases, and, if it is elevated, permanent ventricular drainage should be instituted by means of a device that permits the ventricular fluid to flow off whenever the pressure exceeds a certain height. They resorted to this form of drainage in 28 cases and found that loss of consciousness, severe vomiting, and convulsions often ceased soon after it was instituted. The favorable outcome to this intervention.—*Journal A.M.A.*

PEDIATRICS AT THE TURN OF THE CENTURY

From time to time the Archives, which was the first Children's Journal in the English language, will reprint contributions by the pioneers of the specialty over fifty years ago. It is believed that our readers will be interested in reviewing such early pediatric thought.

ACUTE ALEUKEMIC LEUKEMIA*

REPORT OF CASE IN A BOY TWO AND ONE-HALF YEARS OLD

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Philadelphia.

The notes of this case are as follows: R. V., male, two and one-half years old, was admitted to the Drexel Children's Hospital March 11, 1905, with a negative family history, both parents and all the other children being alive and well. The patient had been breast-fed for the first seven months of life; he had had whooping-cough when three months old and measles at one year; otherwise he had been in robust health until the beginning of the present illness seven weeks previously. At that time his mother was confined to bed for a few days and he received little attention, living in a cold room most of the time; after this he seemed to have something like the gripe, which was followed six weeks later with swelling of the feet and lower eyelids. He had neither headache nor vomiting, but he was constipated and passed a diminished amount of urine, and he became steadily paler.

Condition on Admission. General development, good; marked anemia; lower eyelids so puffy that the eyes are almost closed; considerable hemorrhage under the skin and also under the ocular and palpebral conjunctiva; numerous petechiae on forehead and on cutaneous surface of lips; numerous submucous hemorrhagic spots, 2 mm. in diameter on dorsal and under surfaces of tongue and on buccal mucous membrane; on the abdomen, near the right anterior superior iliac spine is a black-and-blue spot 2.5 cm. in diameter; smaller spots are sprinkled over the abdomen and legs, but not so profusely as on the forehead. The mind was clear.

*Read before the Seventeenth Annual Meeting of the American Pediatric Society, Lake George, N. Y., June 19, 1905.

At the time of writing this paper the author was Visiting Physician to the Children's Hospital and the Methodist Hospital, Philadelphia, Pa.

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there was no retraction of the neck, no exaggeration of the knee jerk, no ankle clonus, and Kernig's and Babinski's signs were negative. The breath-sounds were normal everywhere over the lungs. The heart-action was rapid, but the sounds were clear throughout the course of the case. The liver was palpable, 6 cm. below the costal border and the spleen 3 cm. Only one urinalysis is recorded, on the third day after admission; neither albumin nor sugar being present, the sediment consisting of epithelial cells and leukocytes. The urine was frequently passed in the bed, but it was possible to collect and measure the following daily amounts: 520 cc., 150 cc., 370 cc., 470 cc., 290 cc., 330 cc., so that the kidneys seemed to be acting well with a minimum of 17 ounces on some days, the rest being lost.

The blood count showed red blood corpuscles, 1,390,000; leukocytes, 6,300; hemoglobin, 23 per cent. There was considerable difficulty in stopping the flow of blood from the puncture of the finger, but pressure finally succeeded after about an hour. The result of the blood count led us to fall back on the diagnosis of purpura hemorrhagica, but as the lymphatic glands, axillary and inguinal, became palpable in a few days, and with the result of the differential count of the leukocytes we were led to regard the case as one of acute leukemia of the lymphatic type, with the leukemic manifestation held in abeyance by some unknown condition. A. O. J. Kelly has reported from the laboratory of the German Hospital such a case, in which syphilis was present, and he thinks it possible that the absence of increase in the leukocytes might have resulted from the associated lues. In this case we are at a loss to know what could have been the deterring factor. The differential counts were made both by the resident physicians and myself with striking similar results, a count of 700 leukocytes giving: Polymorphonuclears, 8.85 per cent; large mononuclears, 4.73 per cent; lymphocytes, 85.00 per cent; myelocytes, 1.42 per cent. The stained red corpuscles showed microcytes, macrocytes, poikilocytes and normoblasts.

Four days after admission nose bleed occurred and was followed in a few hours by vomiting of clotted blood. Subsequently the bowel-movements were dark brown in color, but the vomiting after that did not show blood, so it is possible that the intestinal tract was not the seat of hemorrhages.

The temperature ranged between 99.5° and 101° for three days after admission and then remained close to the 101° line for three days more, rising on the seventh day to 105.4°, falling the next morning to 101°, when death occurred from exhaustion. It is to be regretted that an autopsy was not permitted.

As the case stands it is, of course, incomplete and the naming of it is largely a matter of speculation. It was evidently an infection whose manifestations were localized mainly in the blood and lymphatics, and as acute lymphatic leukemia is so considered, and as this form does not necessarily have as high a leukocyte count as the other forms, it seems more reasonable to class the case here than elsewhere. I prefer to consider it as such rather than purpura hemorrhagica, because the bleeding was not the most prominent symptom and because of the differential count. Malignant endocarditis is not absolutely excluded in the absence of an autopsy, but the heart-sounds never showed even the softest murmur; there were no signs of embolism and the kidneys were unaffected.

By a curious coincidence the issue of the *Journal of the American Medical Association* for March 18, 1905, the day the patient died, refers to a case reported by Dr. Geo. Blumer, of acute Hodgkin's disease, the description of which is strikingly like the above, the author remarking that perhaps it would be better to consider it as acute aleukemic leukemia.

UNDESIRABLE SECONDARY EFFECTS OF BCG. (Nederlandsch Tijdschrift v. Geneeskunde, Amsterdam, 96: 1589, June 28, 1952). Keizer describes observations on seven young children from 2 months to 2 years old who were treated with BCG. In the first group (four boys and one girl) the BCG was given by mouth. All five of these children had general lymphadenitis, atypical tuberculous infiltrates, scrophuloderma, multiple breakdown of lymph nodes, tracheobronchial lymphadenitis with atelectasis, otitis media, mastoiditis, and peripheral facial paralysis. The two boys of the second group received the BCG by hypodermic injection. They had chronic ulcerations of the skin and early signs of tuberculous lymphadenitis. All these children were strong and well nourished at the time of treatment with BCG.—*Journal A.M.A.*

BOOK REVIEWS

MODERN CONCEPTS OF COMMUNICABLE DISEASE. By Morris Greenberg, M.D., M.S.P.H. and Anna V. Matz, R.N., M.A. Cloth. Illustrated. Pp. 553. Price \$6.20. New York: G. P. Putnam's Sons, 1953.

This book is divided into two parts, the first of which considers the basic principles of communicable disease. In this section the authors offer a recommendation which should be accepted widely since it is only an old custom which increases the work of the laboratory, viz.: "The newer knowledge of communicable disease indicates that the routine taking of cultures from the throats of all children, and of vaginal smears of all girls admitted to hospitals, institutions, daycare agencies, shelters, or camps should be discouraged." The second part of the book is devoted to the specific communicable diseases. The diseases are considered under four categories depending on the method of transmission, i.e., respiratory diseases, food and water-borne diseases, contact diseases and diseases transmitted by arthropods. The discussion of the various diseases is brief and to the point without any waste of words. The sentences are short and decisive. The value of the book would have been greatly enhanced if some colored photographs of the various typical rashes were included. This is all the more so since the book is obviously intended for the medical student and nurse in training. Very few errors were noted and on the whole the book is a valuable text.

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CLINICAL ORTHOPAEDICS. No. 1. Edited by Anthony F. De Palma, M.D. Cloth. Illustrated. Pp. 242. Price, single issue \$6.00, sustaining \$5.00. Philadelphia: J. B. Lippincott Company, 1953.

This volume introduces a new publication to be published at the present time semiannually. The purpose of the publication, as noted by the editor in the preface, is to bring important orthopedic information of scientific, practical and academic value "not only to orthopaedic surgeons and traumatic surgeons but also to all those in the allied specialties and, above all, to the general practitioner." Each issue is to be divided into two parts, one devoted to a specific topic and the other to miscellaneous articles. The first issue is

devoted to Children's Orthopedics in the first section and the second section is devoted to general orthopedics. If this publication is aimed at the non-orthopedist then the articles chosen should contain a great deal more regarding treatment and treatment instructions should be more specific. This is the only criticism I can offer. The articles are well written, the illustrations good and the format is in good taste and the printing easy on the eyes. Several articles are of special interest and worthy of note: Adolescent Flared Ribs by G. Pitkin, Calcaneovalgus Foot in the Newborn and Its Relation to Developmental Flatfoot by C. F. Ferciot, Structural Scoliosis by R. T. McElvenny and Treatment of Poliomyelitis Based on Pathophysiology by N. M. Shutkin.

MICHAEL A. BRESCIA, M.D.

ADVANCES IN PEDIATRICS. Vol. VI. Edited by S. Z. Levine, M.D. Cloth. Illustrated. Pp. 323. Price \$7.50. Chicago: Year Book Publishers, Inc., 1953.

This last volume of the *Advances* like its predecessors contains several interesting articles and excellent reviews of some subjects. The chapter on the Lipoidoses by S. van Creveld, for example, is an excellent summary and review of the subject containing a great deal of information which is not usually available in the standard pediatric textbooks. This volume contains six other subjects as follows: Lymphosarcoma in Childhood, by H. W. Dargeon; Preventive Prenatal Pediatrics, by T. H. Ingalls; Intestinal Obstruction in the Neonatal Period, by C. E. Koop; Hemolytic Disease of the Newborn, by P. Levine, et al.; Pulmonary Pathology in the Newborn, by E. L. Potter; and Megaloblastic Anemia of Infancy, by W. W. Zuelzer and J. Rutzky. This book is highly recommended.

MICHAEL A. BRESCIA, M.D.



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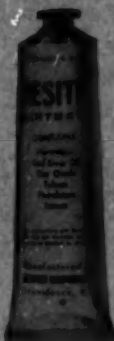


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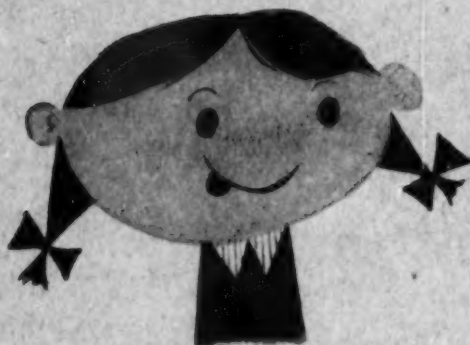
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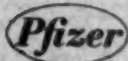
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